CLINICAL PROFILE OF ARRHYTHMIAS COMPLICATING ACUTE ANTERIOR WALL MYOCARDIAL INFARCTION

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CERTIFICATE

This is to certify that this dissertation entitled **"CLINICAL PROFILE OF ARRHYTHMIAS COMPLICATING ACUTE ANTERIOR WALL MYOCARDIAL INFARCTION"** is the bonafide original work of **Dr.P.R.Sowmini** in partial fulfillment of the requirement for MD (Branch I) General Medicine examination of the Tamil Nadu Dr.MGR Medical University to be held in March 2008.

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DECLARATION

I, **Dr.P.R.SOWMINI**, solemnly declare that this dissertation "**CLINICAL PROFILE OF ARRHYTHMIAS COMPLICATING ACUTE ANTERIOR WALL MYOCARDIAL INFARCTION"** is a bonafide record of work done by me in the Department of Medicine, Government Stanley Medical College and Hospital, Chennai under the guidance of **Prof.Dr.V.RUCKMANI, M.D.,** Addl. Prof. of Medicine, Government Stanley Medical College and Hospital, Chennai – 600 001.

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CONTENTS

ABBREVIATIONS

SA node	-	Sino atrial node
AV node	-	Atrio ventricular node
ICCU	-	Intensive coronary care unit
AV block	-	Atrioventricular block
LAHB	-	Left anterior hemi block
LPHB	-	Left posterior hemi block
LBBB	-	Left bundle branch block
RBBB	-	Right bundle branch block
B/L BBB	-	Bilateral bundle branch block
CAST	-	Cardiac arrhythmia suppression trial
LAD	-	Left anterior descending artery
LCX	-	Left circumflex artery
S1	-	I Septal branch
D1	-	I diagonal branch
SHT	-	Systemic hypertension
DM	-	Diabetes mellitus

KEY TO MASTER CHART

DOA	-	Day of admission
I.P.No.	-	Inpatient number
DM	-	Diabetes mellitus
SHT	-	Systemic hypertension
ARR	-	Arrhythmias
R	-	Regular
IR	-	Irregular
S	-	Hemodynamically stable
IS	-	Hemodynamically unstable
TP	-	Temporary pacemaker
ECHO +	-	Echocardiogram with LV dysfunction / RWMA
ECHO -	-	Echocardiogram without LV dysfunction / RWMA
ASMI	-	Anteroseptal myocardial infarction
AWMI	-	Anterior wall myocardial infarction
EAWMI	-	Extensive Anterior wall MI
ALMI	-	Anterolateral MI
ST	-	Sinus tachycardia
SB	-	Sinus bradycardia
SVT	-	Supraventricular tachycardia
AF	-	Atrial fibrillation
CHB	-	Complete heart block
LBBB	-	Left bundle branch block
RBBB	-	Right bundle branch block
BFB	-	Bifascicular block
VT	-	Ventricular tachycardia
VF	-	Ventricular fibrillation
VPD	-	Ventricular premature depolarisation
D	-	Death

INTRODUCTION

Acute Myocardial infarction is the most common and most serious life threatening illness that causes more deaths and disability and incurs greater economic costs than any other illness in the world. The mechanism responsible for infarction related arrhythmias include autonomic nervous system imbalance, electrolyte disturbances, ischemia and slowed condition in the zones of ischemic myocardium.

Arrhythmias have been noted in 70 to 95% of patients with Acute Myocardial infarction. Many arrhythmias may occur prior to hospitalization and thus the overall incidence of arrhythmias in acute myocardial infarction may actually be 100%.

Arrhythmias are responsible for serious hemodynamic disturbances. The most significant consequence of cardiac arrhythmias is fall in cardiac output and B.P. This may precipitate or aggravate congestive cardiac failure. More than 60% of deaths associated with acute myocardial infarction occur within 1 hour of the event and are attributable to malignant arrhythmias usually ventricular fibrillation.

Careful monitoring of cardiac rhythm and prompt treatment of arrhythmias have sharply reduced the incidence of in-hospital deaths from arrhythmias.

Prior to the advent of coronary care units, treatment of acute myocardial infarction was towards healing of the infarct preventing cardiac rupture and management of pulmonary or systemic embolism. Subsequently the major emphasis of the therapeutic strategy is on the prophylaxis and aggressive treatment of arrhythmias.

Lown and his colleagues, through their provocative works, proved that intensive coronary care should be attempted in order to prevent the development of serious ventricular arrhythmias, which serves to reduce the mortality from acute myocardial infarction. This realisation led to the proliferation of mobile coronary care units, ICCU units and intermediate care or step down units.

During the past 3 decades, the mortality of patients with acute myocardial infarction treated in intensive coronary care units has declined from 30% to about 15%. The reduction in mortality has resulted from the elimination of primary arrhythmias as a cause of death. Intensive coronary care units allow continuous monitoring of cardiac rhythm by highly trained personnel, administer immediate support, treatment and prophylaxis of arrhythmias and use specialised cardiac interventions.

Anterior wall Myocardial infarction is the most common presentation of acute myocardial infarction. Left anterior descending artery is the most frequent culprit vessel. The incidence type and severity of arrhythmias varies with the area of the infarct. Tachyarrhythmias are most commonly seen in anterior wall myocardial infarction while Bradyarrhythmias occur frequently in inferior wall myocardial infarction. The overall incidence of arrhythmias and their resultant mortality is more with anterior wall myocardial infarction.

AIM OF THE STUDY

The aim of the study is to analyse :

1. The incidence of different types of arrhythmias in acute anterior wall myocardial infarction in 100 consecutive patients admitted to intensive coronary care unit of Govt. Stanley Hospital.

2. Correlation of each type of arrhythmia with the location and extent of acute anterior wall myocardial infarction.

3. To evaluate the role of age, sex and other risk factors in relation to the various types of arrhythmias.

4. To interpret the prognostic factors for various arrhythmias and their outcome.

5. To assess the effect of arrhythmias on the morbidity and mortality of patients with acute anterior wall myocardial infarction.

REVIEW OF LITERATURE

Anatomy and physiology of the conducting system

The rate and rhythm of the heart are controlled by the *sino-atrial node* (SA node), which is situated in the wall of the right atrium to the right of the superior vena caval orifice. The sinus impulse leaves the SA node and spreads through the atrial muscle; this atrial activation is reflected by the P wave of the electrocardiogram. The sinus impulse eventually reaches the AV node, which is situated in the right atrium above the tricuspid valve and just to the right of the interatrial septum. After a delay at the AV node (reflected in the electrocardiogram as the greater part of the P-R interval) the impulse travels down the bundle of His, bundle branches and Purkinje network system. The *bundle of His* passes horizontally to the left from the AV node, pierces the membranous interventricular septum and divides into right and left bundle branches. These pass down on either side of the muscular interventricular septum and finally divide into the Purkinje network fibres, which proceed vertically to the surface of the heart from the endocardium to the epicardium.

The SA node is mainly under the influence of the vagus nerve, and normal variations in heart rate are effected mainly by variations in vagal tone. An increase in vagal tone slows the heart; a decrease in vagal tone accelerates the heart. The SA node is also influenced to a lesser degree by variations in sympathetic tone.

The pacemakers of the heart

The heart has many potential pacemaking cells. These are situated in the SA node, the AV node, the bundle of His, the atria and the ventricles (every Purkinje cell is a potential pacemaking cell). The SA node has the fastest inherent discharge rate, which usually ranges from 70 to 80 beats per minute. The inherent rate of potential AV nodal pacemaking cells is about 60 beats per minute. The inherent rate of pacemaking cells in the bundle of His is about 50 beats per minute. The inherent rate of the Purkinje cells of the ventricular muscle is about 30-40 heats per minute. In other words, the more distally a potential pacemaker is situated from the SA node, the slower is its inherent discharge rate.

This ensures that there is only one pacemaker which is normally in control of the heart, for, otherwise, chaos would reign supreme. The concept of protection is one of the most important principles governing electrophysiology. Not only does it ensure that there is only one pacemaker governing and in control of the heart, but it permits a slower pacemaker to take over the pacemaking function of the heart when the fastest pacemaker defaults. Thus, if the sinus pacemaker should fail to reach a slower subsidiary pacemaker, due, for example, to marked sinus bradycardia, SA block or AV block, a slower pacemaker in the ventricles will take over the pacemaking function, manifesting as a ventricular escape rhythm.

Classification of arrhythmias[0]

Abnormal rhythms occur as primary and secondary disorders. Primary disorders of rhythm reflect a basic, essential abnormality. Secondary disorders of rhythm only occur as a result of, and secondary to, a primary disorder.

The primary disorders of rhythm

The primary disorders of rhythm may, in simplified form, be classified into two major categories.

1. Disturbances of impulse formation
2. Disturbances of impulse conduction.

Disturbances of impulse formation

Sinus rhythms

Sinus arrhythmia

Sinus tachycardia

Sinus bradycardia

Ectopic atrial rhythms

Atrial extrasystoles

Paroxysmal atrial tachycardia

Atrial fibrillation

Atrial flutter

AV nodal rhythms

AV nodal extrasystoles

Extrasystolic-paroxysmal-AV nodal tachycardia

Idionodal tachycardia

Ventricular rhythms

Ventricular extrasystoles

Extrasystolic ventricular tachycardia

Idioventricular tachycardia

Ventricular flutter

Ventricular fibrillation

Ventricular parasystole

Disturbance of impulse conduction

SA Block

Intra atrial block

Atrio ventricular dissociation

Atrio ventricular blocks

I° AV Block

II° AV Block Mobitz type I AV Block

Mobitz type II AV Block

III° AV Block (Complete heart block)

Bundle branch block

LAHB

LPHB

LBBB

RBBB

B/L BBB

The Secondary disorders of rhythm

(i) Escape rhythms

- Atrial escape
- AV nodal escape
- Ventricular escape

(ii) AV dissociation

(iii) Phasic aberrant ventricular conduction

ARRHYTHMIAS IN MYOCARDIAL INFARCTION

Patients with myocardial infarction can experience a wide range of arrhythmias and conduction abnormalities, from transient and relatively innocuous sinus bradycardia to life-threatening ventricular fibrillation.

Supraventricular tachyarrhythmias (most commonly, atrial fibrillation) generally occur with a rapid heart rate and may cause or exacerbate ischemia, provoke a serious sustained ventricular tachyarrhythmia, or induce or worsen heart failure. However, such arrhythmias are usually not life-threatening.

Ventricular tachyarrhythmias may be asymptomatic and relatively innocuous (eg, ventricular premature beats), asymptomatic but of prognostic importance (eg, nonsustained ventricular tachycardia), or sustained and symptomatic or life-threatening (eg, sustained monomorphic or polymorphic ventricular tachycardia or ventricular fibrillation).

Bradyarrhythmias that result from sinoatrial (SA) nodal dysfunction or from abnormalities of atrioventricular (AV) nodal or His-Purkinje system disease include second-degree and third-degree (complete) heart block.

Etiology of supraventricular arrhythmias

Supraventricular arrhythmias are relatively common in the preinfarction period. Explanations for their frequent occurrence include:

(1) atrial ischemia and, rarely, infarction

(2) stretch and distention of the atrial myocardium resulting from elevated atrial pressure

(3) irritation of the atrial myocardium resulting from pericarditis

(4) elevated sympathetic tone and an increase in circulating catecholamines, and

(5) vagal activation and an increase in parasympathetic tone, especially with an inferior wall infarction.

Sinus bradycardia

Sinus bradycardia occurs in 16% to 25% of patients with acute myocardial infarction, particularly of the inferior or posterior wall[1]. It is most often transient, resulting from an increase in vagal tone. A decrease in myocardial efficiency and a reduction in cardiac output may cause hemodynamic symptoms, including exacerbation of ischemia, reduced mentation, congestive heart failure (CHF), and a more serious escape rhythm.

No specific therapy is needed for asymptomatic sinus bradycardia. If symptoms of hemodynamic compromise or ischemia occur, intravenous atropine sulfate, 0.6 to 1 mg, is usually effective. Persistent bradycardia warrants consideration of temporary pacing, which also may be necessary if the patient requires therapy with drugs that may further depress sinus node automaticity, particularly beta blockers. Since sinus bradycardia usually resolves, long-term pacing is rarely needed.

Sinus tachycardia

About 30% of patients experience sinus tachycardia following myocardial infarction[2]. It represents an appropriate physiologic response to left ventricular dysfunction, CHF, or stimulation and overactivity of the sympathetic nervous system. Treatment therefore is targeted at the underlying cause: angiotensin converting enzyme inhibitors, digoxin and diuretics for CHF and beta blockers for a primary increase in sympathetic output or an increase in circulating catecholamines. The dose is adjusted as the heart rate slows.

Atrial arrhythmias

About 10% to 20% of patients with myocardial infarction experience atrial arrhythmias. These generally occur within 24 hours of infarction[3].

Atrial fibrillation

Atrial fibrillation is seen in 10% to 15% of patients with myocardial infarction[4,6], most commonly in those who have significant left ventricular dysfunction and CHF. The presence of atrial fibrillation in patients with myocardial infarction increases mortality during and after hospitalization (relative risk, 1.7). This increase is due not to the arrhythmia itself but to factors associated with it, particularly left ventricular dysfunction, CHF, and cardiogenic shock[5,6].

Initial treatment of atrial fibrillation depends on the clinical situation and associated symptoms. Often, the arrhythmia is only transient and requires no therapy; even when persistent, it generally responds to appropriate treatment. Heparin therapy should be initiated soon after onset of atrial fibrillation, because anticoagulation reduces the small risk of embolism and eliminates concern about reversion within 48 hours.

Prompt control of the heart rate is essential; an AV nodal blocking agent should be given intravenously--preferably a beta blocker or, if contraindicated, verapamil hydrochloride or diltiazem hydrochloride. Digoxin is not likely to slow the heart rate promptly but may be of use when atrial fibrillation is due to CHF, since digoxin therapy improves left ventricular function and hemodynamics and may result in reversion of atrial fibrillation and restoration of normal sinus rhythm.

Electrical cardioversion to restore sinus rhythm is indicated in patients with severe hemodynamic compromise or refractory symptoms of ischemia due to atrial fibrillation. When atrial fibrillation persists, reversion with antiarrhythmic therapy should be attempted. Although intravenous ibutilide fumarate has been beneficial for reverting new on set atrial fibrillation in about 30% to 40% of patients, its efficacy and safety in patients with myocardial infarction have not been well studied[7]. Its major side effect is torsades de pointes; whether patients who have had a recent myocardial infarction are at increased risk for this complication is uncertain.

Despite concerns raised by the Cardiac Arrhythmia Suppression Trial (CAST) about long-term use of class I antiarrhythmic agents in patients with myocardial infarction, these drugs are often beneficial for acute reversion of atrial fibrillation. In many cases, a single large dose of such a drug results in reversion within 2 or 3 hours[8]. If long-term therapy is necessary, the risks associated with the class I antiarrhythmic agents, particularly the class IC agents, may outweigh the benefits of reversion and prevention of atrial fibrillation. In such cases, sotalol hydrochloride, a drug with class III and beta-blocking activity, is an appropriate first-line agent, since it may prevent atrial fibrillation in patients with myocardial infarction and is safe in this setting[9].

Amiodarone hydrochloride is also very effective for atrial fibrillation and is not associated with increased mortality in patients with myocardial infarction according to the European Myocardial Infarct Amiodarone Trial[10] and the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial[11]. In patients in whom the use of any antiarrhythmic drug is a concern, a reasonable

approach is maintenance of atrial fibrillation with the use of an AV nodal blocking drug (especially a beta blocker) and long-term anticoagulation with warfarin sodium.

Atrial flutter

Atrial flutter is not commonly seen in patients with myocardial infarction, and after reversion it is not likely to recur. As with atrial fibrillation, the first step in management is to slow the heart rate, which can often be achieved with an AV nodal blocking agent. If this is not possible or if atrial flutter is accompanied by symptoms, electric cardioversion is highly effective. An alternative therapy, particularly for asymptomatic atrial flutter, is intravenous ibutilide, which is effective in reverting atrial flutter and restoring sinus rhythm in about 60% of patients.

Since atrial flutter is not likely to recur, long-term suppression is unnecessary. However, if atrial flutter does recur and is accompanied by symptoms, long-term suppressive therapy is indicated. Concern about the use of class I antiarrhythmic agents, particularly class IC drugs, in patients with a recent myocardial infarction has resulted in greater reliance on the class III agents. Use of radiofrequency catheter ablation, which is an effective approach for the prevention and possible cure of atrial flutter, is increasing. However, its use and role in patients with a recent myocardial infarction have not been evaluated.

Etiology of ventricular arrhythmias

Several factors account for the high incidence of ventricular arrhythmias after myocardial infarction:

- Myocardial damage resulting in variable and nonuniform changes in impulse conduction and membrane repolarization
- Hypoxia and acidosis
- Activation of the sympathetic nervous system and resulting sympathetic vagal imbalance
- Spontaneous, mechanically induced, or pharmacologically induced reperfusion, which can result in arrhythmia

Ventricular premature beats of various frequencies are observed in up to 90% of patients with myocardial infarction[12,13]. Nonsustained ventricular tachycardia occurs in up to 40% of such patients, and sustained ventricular tachycardia or ventricular fibrillation each occurs in about 3% to 5%. Mortality in the immediate postinfarction period, as well as during the first year after myocardial infarction, is most often due to sudden death from ventricular fibrillation.

Ventricular premature beats

Ventricular premature beats are usually asymptomatic, and it appears that isolated premature beats themselves in the setting of acute myocardial infarction, regardless of frequency or multiformity, are not associated with an increased risk of sustained ventricular tachyarrhythmia. Therefore, short-term therapy is not recommended unless the frequency of premature beats results in symptoms or hemodynamic instability or triggers more serious arrhythmia. In such situations, lidocaine hydrochloride is the drug of choice. Alternatively, intravenous procainamide hydrochloride may be used.

Long-term antiarrhythmic therapy for ventricular premature beats is potentially hazardous and therefore is not indicated. A number of studies, including CAST[14] and the Survival With Oral d-Sotalol (SWORD) trial[15], have established that such therapy increases mortality.

Ventricular tachycardia

Ventricular tachycardia of several types may be seen in up to 40% of patients after myocardial infarction.

Types: *Accelerated idioventricular tachycardia*, with a rate of 50 to 120 beats per minute, results from either

(1) failure or structural damage of the SA or AV nodal pacemaker or enhanced vagal tone leading to depression of nodal automaticity and function and an escape ventricular focus or

(2) an abnormal ectopic focus within the ventricle, which assumes the role as dominant pacemaker. This arrhythmia most often occurs during reperfusion following thrombolytic therapy, especially when such agents are given early in the course of acute myocardial infarction[16].

When accelerated idioventricular tachycardia results from failure of proximal pacemakers, it represents a stable rhythm. However, when this arrhythmia is due to an ectopic focus, it is potentially unstable; the rate may become rapid, requiring the same therapy as for sustained ventricular tachycardia.

Accelerated idioventricular tachycardia due to an escape rhythm requires no treatment when it is stable and without hemodynamic symptoms. If symptoms are present, atropine therapy may be effective when enhanced vagal tone is the cause, or a temporary pacemaker may be indicated. An antiarrhythmic agent, particularly lidocaine, should not be given, because suppression may result in a profound sinus or junctional bradycardia or even asystole. Sympathetic stimulation with isoproterenol hydrochloride or epinephrine increases the pacemaker rate but has the potential to accelerate the rate in an unpredictable way.

Nonsustained ventricular tachycardia consists of three or more successive ventricular beats lasting up to 30 seconds, with rates greater than 120 beats per minute. *Sustained ventricular tachycardia* lasts more than 30 seconds or is terminated in less than 30 seconds because of hemodynamic compromise. In *polymorphic ventricular tachycardia,* the QRS morphology and RR intervals are variable during each episode, while in *monomorphic ventricular tachycardia*, the QRS complexes are uniform and the RR intervals are fairly uniform, although slight variability may be present.

Management: The approach to evaluation and treatment of ventricular tachycardia depends upon several factors:

(1) Timing: Sustained ventricular tachycardia occurring within 48 hours of myocardial infarction, seen in 2% of patients, is often transient and is not associated with long-term risk of sudden cardiac death[17]. Only short-term suppression (generally 2 days) with an intravenous antiarrhythmic agent such

as lidocaine, procainamide, or bretylium tosylate is required. Amiodarone is frequently effective and may be used after other agents have been tried, particularly if lidocaine or procainamide has been ineffective[18].

When sustained ventricular tachycardia occurs more than 48 hours after acute myocardial infarction in a patient who is otherwise in stable condition and without evidence of CHF or ischemia, aggressive therapy with an antiarrhythmic drug or an implantable cardioverter-defibrillator is required.

(2) Morphology: Polymorphic ventricular tachycardia, seen in 2% of patients with myocardial infarction, is often rapid, symptomatic, and hemodynamically and electrically unstable[19]. It is potentially life-threatening, since it may herald the occurrence of ventricular fibrillation. Polymorphic ventricular tachycardia is commonly associated with underlying and ongoing (overt or silent) ischemia, which warrants the use of anti-ischemic medication or revascularization with either percutaneous transluminal coronary angioplasty or coronary artery bypass grafting if critically stenotic lesions are present. On occasion, placement of an intra-aortic balloon pump is useful. If ventricular tachycardia recurs despite effective control of ischemia, long-term antiarrhythmic therapy or an implantable cardioverter-defibrillator is indicated.

Monomorphic ventricular tachycardia is most often due to a reentrant mechanism and is the result of severe myocardial damage and scar formation. When rapid, symptomatic, or hemodynamically unstable, this arrhythmia requires prompt electric reversion. If ventricular tachycardia recurs despite lidocaine therapy, administration of amiodarone, procainamide, or bretylium can be tried. In some patients, intravenous beta-blocker therapy has been

effective in preventing recurrent arrhythmia, especially if an associated sinus tachycardia preceded the ventricular tachycardia episode.

Overdrive pacing with use of a temporary pacemaker wire has also been of benefit in selected patients. It may be useful for prevention of arrhythmia, especially in the setting of bradycardia due to AV block or sinus node dysfunction.

Long-term therapy is indicated only if the ventricular tachycardia occurs more than 48 hours after the onset of myocardial infarction in a patient who is otherwise in stable condition or if it has been recurrent or difficult to suppress.

(3) Nonsustained versus sustained: Short-term therapy for sustained ventricular tachycardia is often necessary, and long-term therapy for prevention is occasionally indicated. Nonsustained ventricular tachycardia occurring during acute myocardial infarction may indicate a potential for sustained ventricular tachycardia or ventricular fibrillation, but it is uncertain whether prophylactic therapy, aimed at suppression of the arrhythmia, will prevent a sustained episode. In some situations, suppressive therapy with intravenous drugs (lidocaine, procainamide, bretylium, or amiodarone) is indicated; for example, nonsustained ventricular tachycardia that occurs in association with evidence of ongoing ischemia (electrocardiographic [ECG] changes and symptoms), that produces symptoms or hypotension, or that is polymorphic, rapid and frequent, or of long duration. In some patients, intravenous or oral beta-blocker therapy may be effective. Infrequently, overdrive pacing is needed to suppress this arrhythmia.

Therapy for nonsustained ventricular tachycardia that occurs more than 48 hours after the acute event is problematic. Although this arrhythmia is linked to an increased risk of sudden death during the first 6 to 12 months after myocardial infarction[20,21], especially when associated with a reduced left ventricular ejection fraction (<40%), there are no data to support that therapy directed at suppression of nonsustained ventricular tachycardia prevents sudden death[22]. In CAST[14], only 20% of the patients had nonsustained ventricular tachycardia and only 10% had more than one run of ventricular tachycardia in 24 hours.

A proposed approach to management is further risk stratification to identify patients who are truly at high risk (ie, those with multiple risk factors, including positive late potentials on a signal-averaged ECG, absent heart rate variability, QT dispersion on the 12-lead ECG, or ventricular tachycardia induced with electrophysiologic study). Therapy with an antiarrhythmic agent, primarily sotalol or amiodarone, or with an implantable cardioverter-defibrillator should be reserved for such high-risk patients.

Ventricular fibrillation

This rapid, disorganized arrhythmia produces no uniform ventricular contraction, no effective cardiac output, and no recordable blood pressure. The incidence of ventricular fibrillation is highest during the first 24 to 48 hours, particularly within the first 4 hours, after the acute event, and may occur in up to 5% of patients[17]. Prompt cardiopulmonary resuscitation and defibrillation (the only definitive treatment) are essential for survival. Although benefits are

uncertain, antiarrhythmic therapy with lidocaine, procainamide, or bretylium can be administered for recurrent ventricular fibrillation before subsequent shocks are delivered. These agents are usually continued for up to 48 hours after the event. The role of intravenous amiodarone is currently being studied in a number of prospective trials. Intravenous beta-blocker therapy administered at the time of presentation of acute myocardial infarction reduces the incidence of ventricular fibrillation during myocardial infarction[23]. In some patients, intravenous beta-blocker therapy prevents recurrent episodes. The capricorn trial[36] (carvedilol post infarct survival control in left ventricular dysfunction study) proposed that carvedilol suppresses atrial and ventricular arrhythmias in patients with AMI.

Ventricular fibrillation occurring within 48 hours of myocardial infarction is not predictive of higher mortality during the first year after infarction[17]. In contrast, when ventricular fibrillation occurs after 48 hours in a patient who is in clinically stable condition and free of ischemia, CHF, or an electrolyte imbalance, it is considered a primary event, and electrophysiologically guided pharmacologic therapy, empirical amiodarone therapy, or use of an implantable cardioverter-defibrillator is necessary. But prophylactic ICD therapy has not been found to reduce overall mortality in high risk patients with MI[37].

Conduction abnormalities and bradyarrhythmias

Conduction abnormalities and bradyarrhythmias are frequent complications of acute myocardial infarction. They are most often transient,

requiring no specific short- or long-term therapy. When symptoms occur, a temporary pacemaker is important, and if they persist, a permanent pacemaker may be necessary. The availability of external pacemakers has resulted in a decrease in the need for prophylactic pacemakers, especially in view of the complications associated with insertion of temporary electrode catheters. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines[24] for the management of patients with acute myocardial infarction include the indications for therapy for conduction abnormalities and bradycardia.

Abnormalities in SA or AV nodal function or intraventricular conduction may be attributed to one of the following:

- An increase in parasympathetic tone commonly associated with an inferior wall myocardial infarction
- An increase in extracellular potassium, which can cause slowing of impulse conduction
- Local release and buildup of adenosine, a metabolite of adenosine triphosphate breakdown, which slows the velocity of impulse conduction through the AV node
- Ischemia, which results in transient or permanent structural changes of the tissues surrounding the SA and AV junctions

Incidence of AV block

Most of the available data regarding heart block in acute myocardial infarction are from studies performed in the prethrombolytic era, but the incidence of AV block among patients treated with a thrombolytic agent appears to be similar to that among untreated patients.

First-degree AV block occurs in 4% to 14% of patients with acute myocardial infarction. Most often the block is within the AV node.

Mobitz type I second-degree block is observed in up to 10% of patients, but it is usually transient, resolving within 72 hours postinfarction[25,26]. The block is located within the AV node and is most often caused by an increase in vagal tone or, less commonly, ischemia of the AV junction. It is more commonly seen with inferior wall myocardial infarction than with anterior wall involvement.

Mobitz type II second-degree block occurs in less than 1% of patients and usually indicates damage to the AV junction or bundle of His[26]. It is therefore more common with anterior wall infarction. The QRS complexes are usually narrow, but when widened, they reflect concomitant damage to the bundle branches or fascicles and hence more extensive myocardial involvement. Risk of progression to complete heart block is high.

The *2:1 AV block* is a specific form of second degree heart block that may be due to block within the AV node or the His-Purkinje system. It may be either Mobitz type I or Mobitz type II, and this can be established with invasive electrophysiologic study.

Third-degree (complete) heart block is seen in 5% to 8% of patients with acute myocardial infarction and generally occurs early in the course of infarction[26]. Complete heart block is twice as common with inferior or posterior wall infarction as with anterior wall involvement[27].

Incidence of intraventricular conduction block

Intraventricular conduction delays, including bundle branch block and fascicular block, occur in about 10% to 20% of patients with myocardial involvement[28,29]. Up to 22% of patients with a new bundle branch block progress to a high-grade AV block (ie, Mobitz II or complete heart block). Factors associated with an increased risk of complete heart block include a new bifascicular block (right bundle branch block plus left anterior fascicular block, right bundle branch block plus left posterior fascicular block, or left bundle branch block) or a new bifascicular block associated with PR prolongation.

Prediction of complete heart block

The Multicenter Investigation of the Limitation of Infarct Size[30] developed a scoring system to predict the occurrence of complete heart block in patients with acute myocardial infarction. The incidence of new complete heart block was 5.4%, and it occurred a mean of 2.6 days after myocardial infarction. Risk of complete heart block was not associated with the location of the infarction or the left ventricular ejection fraction.

The following factors were considered to be predictive of or associated with increased risk of complete heart block and were each assigned one point:

development of PR prolongation, occurrence of second-degree AV block, left anterior or posterior fascicular block, left bundle branch block, and right bundle branch block. The risk of progression to complete heart block was 1% to 7% with a point score of 0, 8% to 10% with a score of 1, 25% to 30% with a score of 2, and 36% with a score of 3 or more.

Prognosis with complete heart block

The development of complete heart block is associated with a poor prognosis, likely owing to the extensive nature of the acute infarction. In-hospital mortality for both anterior wall and inferior wall infarction is increased by the occurrence of complete heart block, even when other variables are accounted for. In-hospital mortality is significantly higher with anterior wall infarction (63%) than with inferior wall infarction (42%)[27,29]. During the first year of follow-up, complete heart block in the setting of acute myocardial infarction carries a high risk of recurrent AV block and a 28% risk of sudden cardiac death. However, as indicated, the high mortality rate is often due to pump failure from extensive myocardial damage and not bradyarrhythmias, asystole, or tachyarrhythmias.

Non arrhythmic Complications of Acute Myocardial Infarction[30]

Early Complications

1. Failure of reperfusion
2. Left ventricular dysfunction and heart failure
3. Cardiogenic shock
4. Pericarditis

5. Ventricular rupture and ventricular septal defect
6. Acute mitral regurgitation
7. Right ventricular failure

Late Complications

1. Deep vein thrombosis
2. Pulmonary embolism
3. Mural thrombosis and systemic embolism
4. Left ventricular aneurysm
5. Dressler's syndrome
6. Depression

ECG aids in i) Prediction of final infarct size

ii) Estimation of prognosis of and

iii) localization of the culprit vessel.

ECG findings in acute anterior STEMI

Precordial leads

- ST elevation is usually present in V_2 to V_4
- ST elevation in V_4 to V_6 without ST elevation in V_1 to V_3 usually is caused by LCX or distal diagonal occlusion.
- ST elevation in leads V_2 to V_6 may represent LAD occlusion proximal to the first diagonal branch.

Leads I and aVL

- ST elevation in lead I and aVL signifies

1. Occlusion of a short LAD coronary artery before the first diagonal branch (if there is ST elevation in V_2 to V_4)

2. Occlusion of first diagonal branch (if associated with ST elevation in V_2 and isoelectric ST or ST depression in V_3 to V_6)

3. Occlusion of the first marginal branch of the LCX (if there is ST depression in V_2)

- ST depression in aVL signifies LAD artery occlusion distal to the first diagonal branch.

Leads II, III and aVF

- ST depression in the inferior leads signifies

1. Occlusion of a short LAD coronary artery before the first diagonal branch (if there is ST elevation in V_2 to V_4)

2. Occlusion of first diagonal branch (if associated with ST elevation in V_2 and isoelectric ST or ST depression in V_3 to V_6)

- ST elevation in the inferior leads signifies occlusion of a long LAD artery (that wraps the cardiac apex) distal to the first diagonal branch.

Leads aVR

- ST elevation in aVR signifies LAD artery occlusion proximal to the first septal branch.

Right bundle-branch block (new)

- Right bundle-branch block signifies LAD artery occlusion proximal to the first septal branch.

ECG CRITERIA TO IDENTIFY SITE OF OCCLUSION IN THE LAD[32]

Crtierion	Occlusion Site	Sens.	Spec.	PPA	NPA
CRBBB	Proximal to S1	14	100	100	62
ST ↑ V1>2,5 mm	Proximal to S1	12	100	100	61
ST ↑ avR	Proximal to S1	43	95	86	70
ST ↓ V5	Proximal to S1	17	98	88	62
Q avL	Proximal to D1	44	85	67	69
ST ↓ II ≥ 1.0mm	Proximal to S1/D1	34	98	93	68
Q V5	Distal to S1	24	93	71	53
ST ↓ avL	Distal to D1	22	95	87	46
No ST ↓ III	Distal to S1/D1	41	95	92	53

Abbreviations : NPA = Negative predictive accuracy
PPA = Positive predictive accuracy
RBBB = Right bundle branch block.

RISK STRATIFICATION FOR ARRHYTHMIC DEATH AFTER ACUTE ANTERIOR WALL MYOCARDIAL INFARCTION

The process of risk stratification in a patient who has had an acute myocardial infarction has 2 components:

i. Early in-hospital identification of patients at increased risk for recurrent ischemic events.

ii. Identification of patients at increased risk for arrhythmic or nonarrhythmic death.

Risk appears to be equivalent in patients with ST elevation (Q wave) and non ST-elevation (Non-Q wave) infarction.

Risk factors

1. Reduced LVEF (LV ejection fraction)
2. Ventricular tachycardia induced by electrophysiological study (EPS).
3. Spontaneous ventricular premature beats (VPBs) and non sustained (NSVT) ventricular tachycardia documented on 24 hours ambulatory monitoring
4. Late potentials on a signal averaged ECG (SAECG)
5. Reduced heart rate (HRV) variability assessed by ambulatory monitoring
6. T wave Repolarisation alternans (TWA)

Risk stratification[34]

Following recovery from AMI 6 weeks after the event, long term prognosis can be assessed by the following methods:

1. Ambulatory ECG monitoring
2. Exercise testing
3. Signal averaged ECG

SAECG reveals presence of late potentials that are low-amplitude, high-frequency wave forms within the terminal portion of the QRS complexes. Late potentials reflect the presence of slow conduction within the ventricular

myocardium that may serve as a substrate for arrhythmogenesis. The underlying histology is hypothesized to be areas of fibrosis interspersed among areas of viable myocardium. Currently it is well established that β-blockers and successful thrombolysis / revascularization reduce the prevalence of late potentials after acute myocardial infarctions. The prognostic power of late potentials is diminished. With contemporary aggressive treatment for acute myocardial infarction the routine use of SAECG for risk stratification is not recommended.

Clinical implications of the No-Reflow phenomenon[35]

The No-reflow phenomenon occurs after the myocytes in the area are already dead and therefore, later recovery of function is almost impossible. Patients with the above phenomenon form the highest risk subgroup of patients undergoing reperfusion, with raised associated risk of early and sustained congestive heart failure and death.

A large region of no-reflow might impede the ability of the infarct to heal and prevent delivery of pharmacologic agents into that area. Follow up studies have documented that the no-reflow phenomenon is associated with malignant arrhythmias, reduced ejection fraction and a raised risk of cardiac death.

Women with acute myocardial infarction

Women have a 56% excess risk for early mortality after a first transmural MI than men.

ATYPICAL MYOCARDIAL INFARCTION

- Most prevalent in women above 65 years.
- High suspicion index needed for diagnosis
- Common manifestations are

  - Abdominal pain
  - Dyspnoea and
  - Pulmonary edema

- Incidence of complications, both arrhythmic and non arrhythmic is more
- Mortality rate in Atypical MI is higher than that in Typical MI.

MI in elderly

Death rate is elderly patients with AMI was more than that in younger patients with MI.

MATERIALS AND METHODS

100 consecutive patients with acute anterior wall myocardial infarction admitted to the intensive coronary care unit of Stanley Medical College Hospital were studied over a period of 9 months.

The diagnosis of acute myocardial infarction was confirmed with

1. History

Classical chest pain-central retrosternal, constricting, oppressive or burning type of pain usually associated with sweating, often radiating to the inner aspect of the left arm or shoulder or to the root of the neck occurring within 48 hours preceding admission. Patients with h/o prior MI, prexisting valvular heart diseases, prior cardiac surgeries and those who had taken treatment out side were excluded.

2. Electrocardiogram

Electrocardiogram was done in all patients irrespective of symptoms and was considered as the definite evidence of acute myocardial infarction.

3. Bio chemistry

Specific tests CPK-MB-Creatine Phosphokinase fraction specific for myocardial damage was done in all patients Significant levels -->10 IU/L.

Other Tests

Blood urea, Blood sugar and serum creatinine were done for all patients.

SGOT (AST) / serum Aspartate Transaminase level was done for all patients.

Study protocol during Hospital stay

The data regarding the illness of the patients who were admitted was entered into a proforma which included

a. History of symptoms
b. Risk factors
c. Family history and past history of similar illness
d. Physical examination
e. ECG recording
f. Investigations

a. History of symptoms

Angina was diagnosed from the typical history of chest pain.

b. Risk factors

Patients were considered over weight if the BMI-Body Mass Index was greater than 27 kg/m^2 and obese if BMI was more than 30 kg/m^2.

Patients who currently smoke more than 10 cigarettes per day or any number of cigarettes for longer than 6 months were considered as smokers.

Patients were considered hypertensive if the blood pressure recorded was more than 140 mmHg systolic and 90 mmHg diastolic on more than 2 occasions; or evidence of end organ damage such as left ventricular dysfunction, renal function impairment (or) fundus changes suggestive of hypertensive retinopathy were present.

Patient was considered diabetic if the fasting blood sugar value was greater than 126 mg/dl, and post prandial blood sugar was more than 200 mg/dl. History of alcohol intake was elicited; menstrual history was recorded in all females.

c. Family history

Family history of atherosclerotic heart disease was defined as "positive" if any first degree relative of the patient had angina pectoris or myocardial infarction. A history of hyperlipidaemia during admission or raised serum cholesterol above 250 mg% during hospitalisation were considered.

d. Physical examination

A detailed clinical examination including general physical examination, jugular venous pressure and waveforms nature and position of apical impulse were noted. Careful auscultation of the heart was made to note rate, rhythm and other abnormalities.

f. ECG recording

A 12 lead ECG was taken in all patients. The ECG recordings were done immediately on admission, 1 hour after thrombolysis, once daily, whenever complications arise and at discharge.

ECG Evidence was taken as the following

QwMI:

1. ST-T changes with tall peaked "T" waves in hyper acute phase followed by "T" inversion.

2. Deep Q waves more than 0.04 seconds duration and more than ¼ of the height of the following "R" wave in appropriate leads.

NQwMI:

ST segment and T wave changes, chest pain and SGOT/CPK-MB elevations.

Anterior Wall

Anteroseptal – V_1-V_3

Anterior – V_1-V_6

Extensive Anterior Wall-V_1-V_6, 1, aVL

Lateral Wall-V_4-V_5-V_6

High Lateral Wall-1 aVL

Pulse, blood pressure (non-invasive), respiratory rate and rhythm, SaO_2 and ECG were continuously monitored in the ICCU.

Stable patients were transferred to the general medical wards. Stay in ICCU-shortest period was 48 hours and longest period was 12 days. The stay in general ward was 5 days (shortest period) to 15 days (longest period).

ECG criteria for diagnosis of various arrhythmias

1. Sinus tachycardia

Heart rate >100 /mt

Normal 'P' wave

Normal 'PR' interval

2. Sinus bradycardia

Heart rate < 60 /mt

Normal 'P' wave

Normal 'PR' interval

3. Premature Atrial Beats (APD)

i. A 'P' wave that occurs before the next expected series of impulses.

ii. The change in the vector of the early 'P' wave.

iii. The PR for the conducted APD is usually normal (or) minimally prolonged.

iv. Superimposition of the premature P on the T wave of the preceding sinus impulse

v. An unexpected pause due to failure of conduction of a APD to the ventricles.

vi. The hall mark of timing of APD's is the less then fully compensated pause.

vii. Careful inspection of T wave of the sinus impulse before APD will reveal a distortion of the T wave.

4. Supeaventricular tachycardias

- Atrial rate of more than 100 / mt

- Ventricular rate may be less when AV conduction is incomplete

- Narrow QRS complexes

- QRS complexes may be wide if there is aberrant conduction through a bypass tract.

- May be paraxysmal / persistent / chronic.

5. Ectopic atrial tachycardia

- Usually persistent

- P waves are usually normal but not clearly evident when the ventricular rate is rapid.

- With carotid sinus massage, P waves are more evident

6. Multifocal Atrial tachycardia

1. P waves having 3 or more different morphologies that occur in different cycle lengths with marked variations in PP intervals.

2. The rhythm is usually chaotic but rate less than 140/mt

3. Frequent APD present

7. Atrial Flutter

1. A saw tooth pattern in inferior leads.
2. The electrical activity appears continuous without a defined isoelectric line between waves.
3. The most common AV conduction ratios in atrial flutter are 2:1 and 4:1
4. A narrow QRS tachycardia at a rate of 150/mt should always lead to the consideration of Atrial flutter. Carotid sinus massage may be helpful in unconvering atrial flutter.

8. Atrial fibrillation

1. Irregular atrial electrical activity with respect to both rate and rhythm
2. Fibrillatory waves are clearly evident
3. Absent 'P' waves and varying RR intervals

9. AV Junctional Rhythm disturbances

1. Premature AV junctional impulses
2. Junctional rhythm
3. Accelerated junctional rhythms
4. AV junctional tachycardias

The normal inherent rate of AV junctional automatic activity is 40-60/mt. Faster rates from, these levels are considered 'accelerated' rhythms upto 100/mt, after which they are considered 'tachycardia'.

Ventricular Arrhythmias

10. VPD

1. QRS complex is premature, wide and often bizarre in appearance.
2. ST and T wave are opposite in direction to the QRS
3. No premature P wave precedes the premature QRS complex.
4. The pause after VPD is fully compensatory
5. If very premature, 'R' wave of the premature beat occurs on the apex of 'T' of the previous complex (R on T phenomenon).
6. The VPD may be so late that it fuses with the subsequent QRS complex.
7. If the VPD captures the SA node, then the pause will be less than compensatory.
8. The VPD may be interpolated –when the sinus rhythm is slow, the VPD is premature and its retrograde conduction is blocked at the AV node. here is prolongation of PR of the subsequent complex.
9. If the coupling intervals are fixed, then the mechanism is reentry.
10. Coupling intervals can vary markedly when a parasystolic ventricular rhythm is present (or) there is substantial instability in a reentrant circuit (or) when VPD's are multifocal.
11. When every other QRS is a VPD, it is called bigeminy and when every 3^{rd} QRS is a VPD, it is called trigeminy; when every 4^{th} QRS is a VPD, it is called quadrigeminy.

12. 2 successive VPD's are called couplets.

Lown et al. believed that VPC's predispose to ventricular fibrillation and graded the VPC's as follows:

Grade O : No VPC's

I : Less than 30 VPC's / hours

II : More than 30 VPC's / hour

III : Multifocal VPC's

IV-A : Coupled VPC's / Bigminy

IV-B : Salvos of 3 or more (UT)

V : R on T phenomenon

11. Ventricular tachycardia

3 or more VPD's in succession at a rate of more than 140-200 cycles/mt

12. Ventricular capture

If the rate of VT is not very rapid occasional sinus impulses may capture the ventricles producing a normal QRS complex.

Fusion Complex

If the rate of VT is not very rapid a QRS complex intermediate in contour between a normal QRs and that of ventricular tachycardia occurs.

Accelerated indioventricular rhythm

3 or more consecutive QRS complexes of ventricular origin with a rate between 50 and 100 cycles/mt.

Twice as common in inferior infarct; common during Reperfusion therapy.

13. Ventricular parasystole

Automatic rhythm in the ventricles that is reasonably independent of the sinus rhythm.

Varying coupling of VPD's and a common denominator for interectopic intervals.

Ventricular flutter

Ventricular tachycarrhythmias more than 200/mt (usually 240-280/mt) large sinusoidal / zigzag waves present.

15. Ventricular fibrillation

Absence of QRS complexes and T. Waves presence of low amplitude baseline undulations.

CONDUCTION DISTURBANCES

16. SA Block

Simulates sinus bradycardia

Absent 'P' wave and 'QRS' complex intermittently.

17. AV Block

I° AV Block – All atrial impulses are conducted to the ventricles with delay - seen in ECG as prolonged PR intervals.

II° AV Block – Few atrial impulses are conducted to the ventricles. Few are not conducted.

Mobitz type 1 AV block – Wenkebach phenomenon

ECG shows progressive prolongation of the PR interval followed by a missed beat.

Mobitz type 2 AV block

Intermittent missed beats seen in the ECG with no preceding prolongation of PR interval.

III° AV Block

None of the atrial impulses are conducted to the ventricles.

ECG shows complete AV dissociation.

Criteria for Right Bundle Branch Block

Lead V1 Late intrinsicoid, M-shaped QRS (RSR'); sometimes wide R or qR

Lead V6 Early intrinsicoid, wide S wave

Lead 1 Wide S wave

Criteria for Left Bundle Branch Block

Lead V1 QS or rS

Lead V6 Late intrinsicoid, no Q waves, monophasic R

Lead 1 Monophasic R wave, no Q

Criteria for Left Anterior Fascicular Block

1. Left axis deviation (usually ≥ – 60 degrees)
2. Small Q in leads I and aVL, small R in II, III and aVF
3. Usually normal QRS duration
4. Late intrinsicoid deflection in aVL (>0.045 s)
5. Increased QRS voltage in limb leads

Criteria for Left Posterior Fascicular Block

1. Right axis deviation (usually ≥ + 120 degrees)
2. Small R in leads I and aVL, small Q in II, III and aVF
3. Usually normal QRS duration
4. Late intrinsicoid deflection in aVL (>0.045 s)
5. Increased QRS voltage in limb leads
6. No evidence for right ventricular hypertrophy

e. Biochemical investigation

Biochemical investigation done at the time of admission are listed below. They were also repeated as and when necessary; Values considered normal in our study are given below.

1. Urine Analysis	:	Albumin-nil, Sugar-nil
2. Haemoglobin	:	More than 10 mg/dl
3. Blood sugar fasting	:	80-100 mg%
Postprandial	:	100-140 mg %
4. Blood Urea	:	20-40 mg %
5. CPK-MB	:	Upto 10 IU/Lit
6. SGOT	:	Upto 40 IU/Lit
7. Serum Cholesterol	:	150-240 mg %

CHEST X-RAY

X-ray chest PA view was done to look for cardiomegaly, pulmonary congestion and hydrothorax.

At discharge a detailed clinical examination with special reference to pulse rate, blood pressure and general condition of the patient were noted and the ECG repeated.

Patients were reviewed every week for 1 month and thereafter every 15 days at the cardiology out patient department.

ECHO was done for all patients admitted to the ICCU with acute MI and the following parameters studied:

1. Regional wall motion abnormalities
2. Left ventricular systolic and diastolic functions.
3. Left ventricular ejection fraction.
4. Left ventricular dimensions.
5. Presence of left ventricular mural thrombus
6. Presence of LV aneurysm
7. Presence of MR/VSD (and)
8. Presence of pericardial effusion

RESULTS AND OBSERVATIONS

Total number of patients with acute myocardial infarction under study = 100.

Table 1

Arrhythmias in acute AWMI

No. of patients with Arrhythmias	No. of patients without arrhythmias
72	28

72% of the patients with acute anterior wall MI had arrhytmias.

Table 2

Sex distribution of acute anterior wall MI

Male	Female
75	25

Total No. of males with Acute AWMI = 75

Total No. of females with Acute AWMI = 25

Table 3

Sex distribution of Arrhythmias in acute AWMI

No. of males with ARR	% of males	No. of females with ARR	% of females
55	73.33%	17	68%

Total no. of males with acute AWMI with arrhythmias = 55 patients = 73.33% of males with AWMI.

Total no. of females with acute AWMI with arrhythmias = 17 patients = 68% of females with AWMI.

Study shows that males had higher incidence of acute AWMI and the resultant arrhythmias.

Table 4

Age and sex distribution and incidence of arrhythmias

Age group	Incidence of MI (No. of patients)			Incidence of Arrhythmias %		
	Male	Female	T	Male	Female	T
31-40	7	-	7	2 (28%)	-	2%
41-50	21	2	23	16 (76%)	-	16%
51-60	22	10	32	16 (73%)	7 (70%)	23%
61-70	17	7	24	13 (76%)	4 (57%)	17%
71-80	8	6	14	8 (100%)	6 (100%)	14%
	75	25	100	55 (73%)	17 (68%)	72%

Youngest patient recorded - 26 yr. male

Oldest patient recorded – 85 yr. male

The highest incidence of AMI and the resultant arrhythmias was observed in the age group (51-60) yrs. in both males and females.

The incidence of arrhythmias increases with age from 41-80 yrs.

Table 5

Sex distribution in relation to location of MI

Sex	Antero septal	Anterior wall with inferior wall	Anterior wall	Extensive anterior wall	Antero Lateral wall
Male	35	1	26	12	1
Female	9	0	10	4	2
Total	44	1	36	16	3

ASMI was the most common presentation

Symptom analysis

Among 100 patients, 86 were admitted with classical angina and 14 with angina equivalents.

Symptom	%
Chest pain	86%
Radiation to (lt) shoulder	36%
Palpitation	19%
Sweating	29%
Syncope	10%
Dyspnoea	32%

Table 7

Risk factor analysis

Risk factor	%
Smoking	76%
SHT	49%
DM	34%
Alcoholism	46%
Obesity	6%
Hyperlipidemia	6%
Multiple RF	60%
No. RF	4%
Post Menopausal	96%

76 patients with acute AWMI had smoking as RF. All of them were males.

Table 8A

Multiple risk factors

Multiple	%
Smoking + Alcohol	40%
DM+SHT+Smoking	10%
HT+Smoking+Alcohol	10%

% of patients with acute AWMI having multiple RF = 60%.

Table 8B

Single RF %	Multiple RF %
40%	60%

Of the single RF, smoking was the most common (76%)

Table 9

Multiple RF in relation to prevalence of Arrhythmias

% with multiple RF	% With Arrhythmias	% without Arrhythmias
60%	96.7%	3.3%

Incidence of arrhythmias was more common in patients with multiple RF.

Table 10

Distribution of arrhythmias in patients with acute AWMI

S.No.	Arrhythmia	Number of Cases
1.	Sinus Tachycardia	12
2.	Sinus Bradycardia	1
3.	SVT	3
4.	Atrial Ectopics	1
5.	Atrial Fibrillation	4
6.	Ventricular ectopics	35
	UC (uncomplicated)	13
	C (complicated with VT,VF)	22
7.	Complete Heart Block	4
8.	SA Block	0
9.	RBBB	3
10.	LBBB	4
11.	RBBB + LAHB	5
12.	No. Arrhythmias	28

VPD's were the most common arrhythmias (35 cases) complicated by VT/VF in 22 cases.

Table 11

S.No.	Site of Infarction	%
1.	Antero Septal	44%
2.	Anterior Wall	36%
3.	Extensive Anterior Wall	16%
4.	Antero Lateral Wall	3%
5.	High Lateral	0%
6.	Inferior + Anterior Wall	1%

Among the site of infarction, anteroseptal was the most common.

Table 12

Sex distribution of arrhythmias

Incidence	Total %	Male %	Female %
Sinus Tachycardia	12	10	2
Sinus Bradycardia	1	1	-
APD	1	1	-
SVT	3	2	1
Atrial Fibrillation	4	2	2
VPD	13	12	1
VT	14	10	4
VF	8	6	2
Complete heart block	4	4	-
RBBB	3	2	1
LBBB	4	2	2
LAHB	-	-	-
RBBB+LAHB	5	4	1
No Arrhythmias	28	20	8

Table 14

Incidence as per Western Literature (Ref. 1)

Sl.No.	Arrhythmia	%
1.	Sinus bradycardia	40%, 1st Hr., 20% 4 Hrs.
2.	Sinus tachycardia	41%
3.	Atrial ectopics	50%
4.	PSVT	2-5%
5.	Atrial flutter	2-3%
6.	Atrial fibrillation	10-15%
7.	Junctional rhythm	1-2%
8.	Ventricular Ectopics	57%
9.	Ventricular tachycardia	10-40%
10.	AIVR	8-20% (I 2 days)
11.	VF	4-18%
12.	Ventricular Asystole	1-14%
13.	I° AVB	14%
14.	II° AVB	4-10%
15.	III°AVB	5-8%
16.	Intraventricular Blocks	10-20%
17.	LAHB	3-5%
18.	LPHB	1
19.	RBBB	2
20.	LBBB	2
21.	RBBB+LAHB	5
22.	RBBB+LPHB	1

Table 15

Pathological types of MI

No. of cases	QMI	NQMI
100	68	32

Table 16

Percentage of Arrhythmia in QwMI and Non QwMI

Total No. of patients with Arr.	No. of patients with Arr.		% of Arr.	
	QMI	NQMI	QMI	NQMI
72	52	20	72%	28%

Table 18

Percentage of Mortality in relation to location of MI

Total Mortality	Ext. Anterior wall		ASMI		AWMI	
	No. of cases	%	No.	%	No.	%
21	16	76%	3	14%	2	10

Table 19

Mortality in acute AWMI

Total no. of deaths	Arrhythmias	Cardiogenic shock
21	19	2

Table 20

Effect of arrhythmias on hemodynamic stability/mortality

Type of arr.	No. of patients	Death
VT/VF	22	20
AF	4	1
CHB	4	-
BBB	12	-
Total	42	21

Table 21

Thrombolysis in MI

Total No. of patients with Acute AWMI	**100**
No. of patients thrombolysed	72
No. of patients not thrombolysed	28

Table 21

Incidence of Arrhythmias in relation to the duration after onset of symptoms

Time (hrs)	**Acute AWMI**	**Acute AWMI with ARR**	**%**
0-1 hrs	26	22	84
1-2 hrs	26	20	76
2-4 hrs	32	24	75
4-6 hrs	11	5	45
> 6 hrs.	5	1	20
Total	**100**	**72**	

The patients coming in the initial hours had a higher incidence of arrhythmias.

Table 22

Sex distribution of mortality

Sex	**Deaths**
Males	12
Female	9
Total	21

DISCUSSION

The findings of our study corroborate well overall with several other studies conducted in different parts of the world regarding etiological factors, symptomatology, risk factors, clinical features, complications and outcome though there are several interesting points of difference in many of these areas, which may be attributed to differences in racial, geographical distribution of the sample studies as well as to the constraints in facilities available and time window when they reach the hospital.

In a study conducted by the Italian University in 1994, it was found that 32% of women above 65 kgs presented with atypical angina of which abdominal pain, dyspnoea and pulmonary edema were the most frequent symptoms[39].

In our study of 100 cases, 25% were women, all of whom were aged above 40 years, 25% of them presented with atypical angina (angina equivalents) of which dyspnoea was the most frequent symptom.

In a study conducted by Sri Jayadeva Institute of Cardiology, Bangalore (1994)[40], women formed 17% of total MI cases, of which 93% were above 40 years.

Infarction occurred mostly (96%) in post menopausal period in our study.

Mortality rate was high (36%) in women. When compared to men (16%), the commonest cause being arrhythmias. The percentage of arrhythmias in females was significantly less (68%) compared to that of males (73%).

The facts brought out from our study have general agreement on a multifactorial etiology of ischemic heart disease and that incidence of the disease increases with age.

Percentage of patients with multifactorial etiology was 60%, 95% of the above patients had arrhythmias, the most common being VPD's.

Dolder et al. (1975)[41] conducted an international study on myocardial infarction in Young men. They found that the cause for high incidence of MI in the young was the high prevalence of risk factors particularly smoking and hyperlipidemia in the west and eigarette smoking and hyperglycaemia in India.

We also found that the most important risk factors prevalent among the younger age groups were eigarette smoking, hypertension and hyperglycaemia.

The incidence of NQMI in our study was 32% and QMI 68%. The long term outcome of NQMI Vs QMI did not differ according to a study by Tokushima's cardiology department (1994)[42]. Long term outcome and complications could not be followed up in our study due to the time frame of study and poor patient compliance. However mortality in ICCU was more in QMI (90%). When compared to NQMI (10%).

Regarding immediate outcome and complications, there was a total 21% mortality within the first 2 wks of MI, about 50% of this occurring in the first 6 hours. VF was the leading cause of death in AWMI.

Compared to the Western study, our mortality rate is still high, partly due to the limited facilities for coronary care and partly due to the delay in bringing the patients to the ICCU after Acute MI. Only 52% reached hospital in the first 2 hours.

Hospital mortality was high among patients aged above 50 yrs in both males and females, compared to those below 50 hrs of age. This observation is in agreement with the findings of Gregory et al. 1983[43].

Bigger et al. 1978[44] and Taylor et al. 1980[45] indicated that the extent of left ventricular damage is a major determinant of survival in patients following Acute MI. We also found that the mortality rate was high when the extent of infarction was more. 70% mortality was in extensive AWMI.

Incidence of VT was 14% as compared to the 10% in the study of Bell et al. 1972[46]. Incidence of VF was 8% in our study as compared to 10% according to Kertes et al. 1984 and 3-5% reported by Garg et al.[47]

Garg et al. (1984)[47] believed that when there was extensive infarction, there was greater chance for the reentry mechanism to operate, resulting in the genesis of Arrhythmias.

Incidence of VPC's (both complicated and uncomplicated) were found to be 35% in our study compared to 36% in textbook of cardiology by Braunwald,[48] 57% reported by Arthur more and 70% by Morrison et al.[49]

Completed heart block was observed in 4 patients out of 100 cases which is consistent with 5% suggested by Braunwald.

Incidence of sinus tachycardia was 12% in our study, as compared to the study of Lours et al. 1967[50], Jewitt et al. 1967[51] which suggest an incidence of 30%.

Regarding adjunctive medical therapy for acute MI, Trauma medical centre, Kansas[52] (1994) advocated oxygen, morphine, nitrates, beta blockers, ACEI and Aspirin. IV heparin and magnesium therapy have been advised in special subsets of the patients thrombolysed with IV streptokinase 75% of them developed AIVR, 20% had S.bradycardia & 5% had conduction defects.

CONCLUSION

- Incidence of Myocardial infarction increases with age.
- Incidence of Myocardial infarction is significantly more in men (75%) compared to that in women (25%).
- In women, Acute MI was observed mostly (96%) in the post menopausal group
- Smoking was the most common Risk factor (76%).
- Hypertension and Diabetes mellitus were the most important risk factors in females.
- Atypical chest pain was more common in females, of which dyspnoea was the most frequent.
- Mortality was higher in older patients
- Cardiac failure and cardiogenic shock were associated with poor prognosis.
- Most common arrhythmia in our study was VPD's
- Arrhythmias are more common in males (73%) against (68%) in females.
- Arrhythmias are more common in those over 45 yrs (97%). The tachyarrhythmias had a higher incidence.

- ❖ Arrhythmias are more common with multiple risk factors (96%).
- ❖ Most benign arrhythmia was occasional VPD's.
- ❖ Most dangerous arrhythmia was VF
- ❖ Atrial flutter, parasystole and I & II° AVB were not recorded seen in our study.
- ❖ Total mortality was 21% of which death was more common in QMI (90%) and Non QMI (10%).
- ❖ Extent and the size of infarct had a direct correlation to mortality

The present study has enabled us to identify the high risk group for observation and follow up.

Risk stratification helps us in early identification of complications and its treatment. The judicious use of interventional surgical and non surgical approaches with life style modification may pave way for revolutionary changes in mortality and morbidity in patients with arrthymias in acute anterior wall MI.

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ETHICAL COMMITTEE APPROVAL

Ref.No. /ME1/2007

Stanley Medical College,
Chennai-1 Dt. -9-2007

Sub: Medical Education – Stanley Medical College, Chennai – Ethical Committee constituted for approval of Dissertation/ Thesis submitted – regarding.

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The Ethical Committee meeting was held on 3-9-2007 and 7-9-2007 to discuss the paper submitted for Dissertation / Thesis.

The following Members of the Ethical Committee were present and discuss in detail for the approval of the papers presented by the individual by means of power point presentation.

Dr.A.Sundaram, Dean incharge,
Dr.S.Madhavan, Prof. of Pharmacology,
Dr.Thenmozhivalli, Prof. of Microbiology,
Dr.S.Natarajan, Prof. of Medicine,
Dr.K.Balasubramanian, Prof. of Physiology,
Dr.M.L.Shyamala, Prof. of Surgery,
Thiru M.Panneerselvam, Junior Administrative Officer.

LIST OF PAPERS SUBMITTED FOR ETHICAL COMMITTEE APPROVAL
ETHICAL MEETING

Dr. Kiruba Mohan, Prof. of Dermatology

1."N.O.C. for PMS study of pregabalin" - Dr.Parimalam Kumar
2. " A Phase IIb/III trial of LLL-3348 of lupin ltd in plaque psoriasis - Dr.A.Ramesh

Dr.M.Thirunavkarasu, M.D.(Psy)D.PM , Prof. of Psychiatry

"Prevalence, socio-demographic variables and method of suicide among various causes of death."

(2)Psychological autopsy of suicide.

V.Rohit
Effect of chewing gums (XYLITOL)

K.Chinthnidhi
Mycotic infections in immuno compromised and cancer patients.

Malavika Prasad
Profile of Hypertensive emergencies - A study of 100 cases from Dept. of medicine, GSH.
~~~~~~~~~~

3. Sandhya Rani.C Final MBBS,

Assessment of coverage and quality of maternal and child health servic at Minjur Primary Health Centre; Block level

4.C.Muralidharan, Final year.

The implications of mobile phones on hearing loss.

5.V.Sarath Chander, 3rd MBBS

Prevalence of Deafness in children.

6.B.Madhusoothanan, 3rd year

(1) Lung functions in type 2 diabetes.

(2)Hyponatremia in intensive medical care patients in GSH.

7.S.Sathyapriya - II MBBS.,

"A study about screening tests for cases of urinary tract infections (UTIs)Using Urine samples."

8.S.Moogaambiga,

"Estended spectrum beta lactamase producing microbes.

POST GRADUATES

1.Dr.R.Arunprakas -M1. P.G.

Analysis of clinical profile of systemic lupus erythematosus

2.Dr.S.Murugananth - M.2 P.G.

Clinical Profile of infectious fevers

3.Dr.N.Loganathan - M2 P.G.

Clinical and Epidemiological profile of Human Leptospirosis in North Chennai.

4.Dr. K. Babu - M3 - P.G.

Study of Clinical Profile of patients with acute inferior wall myocardial infarction.

5.Dr. S.P.Maharajan - M3 - P.G.

Analytical study of atrial fibrillation in Govt. Stanley Medical College Hospital.

6.Dr.P.R.Sowmini - M3 - P.G.

Clinical profile of arrhythmias complicating acute anterior wall myocardial infarction.

7.Dr.E.Uma Maheswari - M4 - PG

Clinical Radiological analysis of Focal seizures with CT Scan.

8.Dr.S.Sudha Selvi, M4 - PG

Clinical profile of chronic obstructive pulmonary disease.

9.Dr.N.Jayanthi. M6- PG

Prevalence of B2 glycoprotein 1 Dependent anticardiolipin antibodies in acut ischemic stroke.

10.Dr.Lavanya. S. - MD PG

Comparative study of fasting lipid profile in chronic renal failure patients on conservative management, on dialysis and after renal transplant.

11.Dr.R.Geetha - Pharmacology

Evaluation of the sedative effects produced by antihistamines inhealthy volunteers by new techniques.

12.Dr.K.G.Devibala, Pharmacology

To evaluate the efficacy of rupatadine in controlling pruritis in lichen planus.

13.Dr.B.Anitha, Physiology

Visual Evoked potentials in hypothyroid patients.

14.Dr.M.Thirumaran, Physiology

Heart rate variability analysis in alcohol dependant individuals.

15.Dr.K.Vinod, Anaesthesia

Real time ultra sound guided catheterization of IJV – A prospective comparison with land mark guided technique.

16.Dr.Rajesh. C.P. – M6 – PG

Cardiac conduction abnormalities and asymptomatic myocardial infarction in NIDDM patients.

The papers presented to the Committee members by the Profs./Asst. Prof./Post Graduates/Under graduates were discussed across the table while their presentation.

The above papers discussed in detail with its supportive documents submitted by them and approved the above papers submitted for Ethical Committee.

Name of the Members	Signature
Dr.A.Sundaram, Dean incharge,	
Dr.S.Madhavan, Prof. of Pharmacology,	8/10/07.
Dr.Thenmozhivalli, Prof. of Microbiology,	
Dr.S.Natarajan, Prof. of Medicine,	9.10.2007.
Dr.K.Balasubramanian, Prof. of Physiology,	
Dr.M.L.Shyamala, Prof. of Surgery,	08/10/07
Thiru M.Panneerselvam, Junior Administrative Officer.	

PROFORMA

Sl.No.

Name

Age

Sex

DOA

DOD

Occupation

IP No.

SYMPTOMATOLOGY

Angina

Radiation

Sweating

Palpitations

Breathlessness

Syncope

RISK FACTORS

Obesity

IHD

DM

SHT

Smoking

Alcoholism

H/o. CVA

OCP

Menopausal

Hypercholesterolemia

FAMILY HISTORY

IHD

DM

SHT

CVA

SCD

ON EXAMINATION

PR

Rhythm

BP

Temp C

Anaemia

Cyanosis

Sweating

PE

Markers of Atherosclerosis

JVP

CVS PALPATION

AI

Thrill

Palpable sounds

CVS AUSCULTATION

S1

S2

S3

S4

Murmurs

Rub

RS

Added Sounds

ABDOMEN

FF

Organomegaly

CNS

INVESTIGATIONS

Se.Cholesterol

SGOT

CPK-MB

CRP

Urea

Sugar

Creatinine

ECG

CXR

ECHO

TREATMENT

Thrombolysed

Nasal O2

Sedation

Anticoagulants

Anti failure Drugs

Anti Arrhythmics

Vaso dilators

Others

Defibrillation

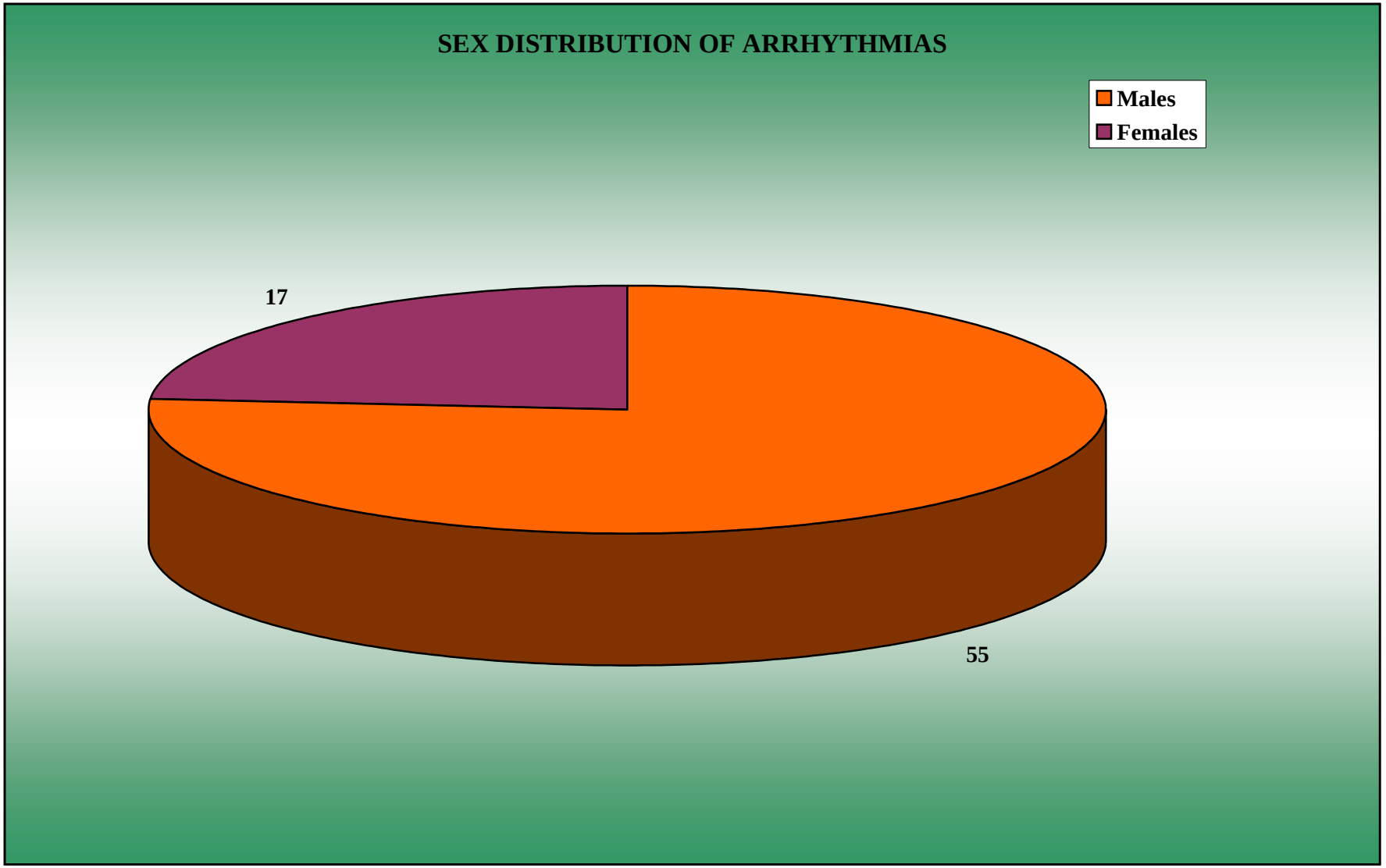
MASTER CHART

Sl.No.	Name	Age	Sex	DOA	Time of presentation (Hrs.)	IP No	SYMPTOMATOLOGY					RISK FACTORS								ON EXAMINATION						TREATMENT		
							Angina	Sweating	Palpitations	Breathlessness	Syncope	Obesity	IHD	DM	SHT	Smoking	Alcoholism	Menopausal	Hypercholesterolemia	PR	Rhythm	BP	ECG	ARR	ECHO	Thrombolysed	Anti Arrhythmics	Defibrillation
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	26	28
1	Manga Lakshmi	65	F	5.1.07	2	528	-	+	-	+	-	-	+	-	-	-	-	+	+	90	R	S	ASMI	VPD	+	-	-	-
2	Sulochana	70	F	16.1.07	4	1520	-	+	+	+	-	-	+	+	-	-	-	+	+	142	IR	S	ASMI	AF	+	-	+	-
3	Nataraj	52	M	16.1.07	3	1590	+	-	-	-	-	+	-	-	+	+	+	-	-	96	R	S	EAWMI	RBBB	+	+	-	-
4	Govindaraj	42	M	16.1.07	1	1598	+	-	-	+	-	-	-	+	+	+	+	-	-	80	IR	S	ASMI	VPD	+	+	+	-
5	Abdul Razak	48	M	17.1.07	4	1697	+	+	+	-	-	+	+	-	+	+	+	-	-	112	R	IS	ASMI	ST	+	+	-	-
6	Komalavalli	55	F	17.1.07	3	1743	-	+	+	+	-	-	+	+	-	-	-	+	-	80	R	IS	EAWMI	LBBB	+	+	-	-
7	Selvaraj	40	M	22.1.07	2	2269	+	-	+	+	-	-	-	-	+	+	+	-	-	30	IR	S	AIMI	CHB	+	+	TP	-
8	Kuppam	50	M	27.1.07	4	2826	+	-	-	-	-	-	-	-	+	+	+	-	-	96	R	S	ASMI	ST	+	+	-	-
9	Janaki	60	F	2.2.07	4	3305	+	+	+	+	-	-	+	+	+	-	-	+	+	146	R	S	AWMI	SVT	+	+	-	-
10	Ragaiah	74	M	3.2.07	7	3694	+	-	-	+	-	-	-	-	+	+	+	-	-	98	R	S	AWMI	BFB	+	+	-	-
11	Arunachalam	65	M	4.2.07	6	3740	+	-	-	+	+	-	-	-	+	+	+	-	-	90	R	S	ALMI	VPD	+	+	-	-
12	Shankar	45	M	9.2.07	3	4371	+	+	-	-	-	-	+	-	+	-	-	-	-	88	IR	S	AWMI	VPD	+	+	+	-
13	Loganathan	64	M	10.2.07	5	4508	+	-	+	+	-	-	-	+	+	+	+	-	-	128	R	S	ASMI	SVT	+	-	+	-
14	Nasariah	60	M	12.2.07	4	4684	+	-	+	-	-	-	-	+	+	+	+	-	-	80	R	S	ASMI	-	+	-	-	-
15	Mariyappan	62	M	15.2.07	1	5070	+	-	-	+	+	-	-	-	+	-	-	-	-	74	R	S	AWMI	VPD	+	-	+	+
16	Kayanchi	55	M	17.2.07	2	5376	+	-	-	-	-	-	-	+	+	+	+	-	-	140	R	IS	AWMI	-	+	-	-	-
17	Bhavani Dasan	46	M	22.2.07	4	5835	+	-	+	-	-	+	-	-	+	+	+	-	-	80	R	S	ASMI	VPD	+	+	-	-
18	Karpagaraj	53	M	22.2.07	2	5950	+	-	+	+	-	+	-	+	+	+	-	-	+	96	R	S	ASMI	VT	+	+	-	-
19	Mohan Kumar	47	M	24.2.07	5	6087	+	-	-	+	-	-	-	-	+	+	-	-	-	82	R	S	EAWMI	BFB	+	+	-	-
20	Munusamy	54	M	24.2.07	3	6070	+	-	-	-	-	-	-	-	+	+	+	-	-	84	R	S	AWMI	-	+	+	-	-

21	Shahul Hameed	66	M	26.2.07	6	6334	+	-	-	+		+	-	+	+	-	-	-	-	106	R	S	AWMI	ST	+	+	-	-
22	Umar Jahn	58	M	27.2.06	4	6404	+	-	-	-	+	-	-	-	+	+	+	-	-	111	R	S	EAWMI	ST	+	+	-	-
23	Vella Thai	60	F	28.02.07	2	6638	-	-	+	+	-	-	-	+	-	-	-	+	-	120	R	S	AWMI	VT	+	-	+	+
24	Govindammal	85	F	2.03.07	2	6804	-	+	-	-	-	-	+	-	-	-	+	+	+	116	R	S	AWMI	VF	+	-	+	+
25	Narayanasamy	70	M	9.03.07	3	7633	+	-	+	-	+	-	-	+	+	+	-	-	-	144	R	S	ASMI	VT	+	+	+	+
26	Venkatesan	42	M	10.03.07	4	7818	-	+	-	+	-	-	+	-	-	+	-	-	-	68	R	S	ASMI	-	+	+	-	-
27	Mohan Kumar	75	M	10.03.07	1	7908	+	-	-	-	-	+	-	-	+	+	+	-	-	146	R	S	ASMI	SVT	+	-	+	-
28	Joseph John	48	M	12.03.07	40 min	8122	+	-	-	+	-	-	-	+	+	+	-	-	-	74	R	S	AWMI	-	+	+	-	-
29	Tharamani	52	F	16.03.07	1 hr.	8597	+	-	-	+	-	-	-	-	+	-	-	+	-	154	IR	S	ASMI	AF	+	+	+	-
30	Kalyana Sundaram	54	M	18.03.07	2 hr.	8745	+	+	+	-	-	-	+	+	+	+	+	-	-	88	R	S	EAWMI	-	+	+	-	-
31	Krishnan	70	M	28.3.07	3	10025	-	+	-	+	-	+	+	-	-	+	+	-	-	80	R	S	ASMI	LBBB	+	-	-	-
32	Venkataiah	65	M	28.3.07	2	10026	-	+	+	+	-	-	+	+	-	+	-	-	-	76	R	S	ASMI	-	+	-	-	-
33	Elizabeth	60	F	30.3.07	3	10286	+	-	-	-	-	-	-	-	+	-	-	+	-	80	R	S	AWMI	-	+	+	-	-
34	Deivasigamani	65	M	30.3.07	1	10523	+	-	-	+	-	-	-	-	+	+	+	-	-	140	R	S	ASMI	VT	+	+	+	+
35	Rajeshwari	65	F	1.4.07	5	10528	+	+	-	-	-	-	+	+	+	-	-	+	-	96	R	S	AWMI	-	+	-	-	-
36	Raghupathy	70	F	2.4.07	2	10584	-	+	-	+	-	-	+	-	-	+	-	-	-	136	R	S	ASMI	VF	+	+	+	+
37	Purushothaman	56	M	13.4.07	6	11845	+	-	-	+	-	-	-	+	+	+	-	-	-	101	R	S	ALMI	ST	+	-	-	-
38	Pooammal	65	F	13.4.07	8	11946	+	-	-	-	-	-	-	-	+	-	+	+	-	110	R	S	AWMI	ST	+	+	-	-
39	Ramammal	60	F	14.4.07	1	12959	+	+	+	-	-	-	+	+	+	-	-	+	+	124	R	S	EAWMI	VF	+	+	+	+
40	Ramu	50	M	21.4.07	1	12974	+	-	-	-	-	-	-	-	+	+	+	-	-	80	R	S	AWMI	-	-	+	-	-
41	Yuvaraj	41	M	23.4.07	2	13068	+	-	-	-	+	-	-	-	+	-	-	-	-	82	R	S	AWMI	SB	-	-	-	-
42	Rukmaniammal	64	F	27.4.07	3	13525	+	+	-	-	-	-	+	-	+	-	-	+	+	110	R	S	EAWMI	VT	+	+	+	+
43	Munusamy	40	M	27.4.07	2	13526	+	-	+	+	-	-	-	+	+	-	+	-	-	80	R	S	EAWMI	-	+	+	-	-
44	Sampath	70	M	27.4.07	1	13565	+	-	+	-	-	-	-	+	+	+	-	-	-	90	R	S	ASMI	-	+	+	-	-
45	Pushpammal	80	F	4.5.07	1	16363	+	-	-	+	+	-	-	-	+	-	-	+	-	94	R	S	EAWMI	-	+	-	-	-
46	Ramalingam	63	M	4.5.07	1	14378	+	-	-	-	-	-	-	-	+	+	+	-	-	106	R	IS	ASMI	VT	+	-	+	+
47	Maliga Behum	54	F	4.5.07	1	14442	+	-	-	-	-	-	-	-	+	+	+	-	-	90	R	S	ASMI	VF	+	-	+	+
48	Selvam	43	F	5.5.07	3	14842	+	-	-	+	-	-	-	-	+	+	-	-	-	94	R	S	EAWMI	ST	+	+	-	-
49	Thangaraj	37	M	9.5.07	4	15084	+	-	-	+	-	-	-	+	+	+	-	-	-	88	R	S	EAWMI	VPD	+	+	-	-
50	Saradha	75	F	11.5.07	2	15295	+	-	-	-	-	-	-	-	+	-	+	+	-	90	R	IS	ASMI	-	+	-	-	-

51	Raja Kahn	45	M	12.5.07	5	15371	+	-	-	-		-	-	-	+	-	-	-	-	96	IR	S	ASMI	VPD	+	+	-	-
52	Karunakaran	50	M	13.5.07	1	15606	+	-	-	-	+	-	-	-	+	+	-	-	-	90	IR	S	ASMI	APB	-	-	-	-
53	Ramachandran	55	M	13.5.07	2	15830	-	-	+	+	-	-	-	+	-	+	-	-	-	114	R	S	AWMI	-	-	-	+	-	-
54	Natarajan	65	M	15.5.07	3	15920	-	+	-	-	-	-	+	-	-	+	+	-	-	88	R	S	ASMI	ST	+	+	-	-
55	Manickam	55	M	15.5.07	3	15923	+	-	+	-	+	-	-	+	+	-	-	-	-	112	IR	S	ASMI	VPD	+	+	+	+
56	Syed Basha	74	M	15.5.07	2	16173	-	+	-	+	-	-	+	-	-	-	-	-	-	80	R	S	ASMI	ST	+	+	-	-
57	Santhosh	49	M	17.5.07	3	16423	+	-	-	-	-	+	-	-	+	+	+	-	-	78	R	S	EAWMI	-	+	+	-	-
58	Ramamoorthy	57	M	10.5.07	3	16521	+	-	-	-	-	-	-	-	+	-	-	-	-	136	R	S	AWMI	AF	+	+	+	+
59	Ezhumalai	50	M	21.5.07	3	16910	+	-	-	+	-	-	-	-	+	+	-	-	-	80	IR	S	AWMI	-	+	+	-	-
60	Ravi	53	M	23.5.07	4	16926	+	+	+	-	-	-	+	+	+	+	-	-	-	150	R	S	ASMI	VT	+	-	+	+
61	Poonusamy	63	M	25.5.07	2	17118	+	-	-	-	-	-	-	-	+	+	+	-	-	80	R	S	AWMI	VPD	+	+	-	-
62	Ramamoorthy	57	M	25.5.07	1	16521	+	-	-	-	+	-	-	-	+	+	-	-	-	130	R	S	ASMI	VF	+	-	+	+
63	Velayudham	65	M	25.5.07	3	17192	+	+	-	-	-	-	+	-	+	+	+	-	+	80	R	S	EAWMI	VPD	+	+	-	-
64	Bee Bee John	80	F	26.5.07	3	17236	+	-	+	+	-	-	-	+	+	-	-	+	-	112	R	IS	ASMI	VT	-	-	+	+
65	Shanthi	50	F	31.5.07	6	17828	+	-	+	-	-	-	-	+	+	-	-	+	-	70	R	S	ASMI	-	-	+	-	-
66	Narayanan	44	M	4.6.07	5	18275	+	-	-	+	+	-	-	-	+	+	-	-	-	68	R	S	EAWMI	-	+	+	-	-
67	Soundarajan	47	M	10.6.07	4	18442	+	-	-	-	-	-	-	-	+	+	+	-	-	70	R	S	ASMI	VPD	+	+	-	-
68	Mangalamary	60	F	14.6.07	3	19125	+	-	-	-	-	-	-	-	+	-	-	+	-	110	R	S	AWMI	ST	+	+	-	-
69	Dhanapal	26	M	16.6.07	2	19751	+	-	-	+	-	-	-	-	+	+	-	-	-	80	R	S	ASMI	-	+	+	-	-
70	Manohar	53	M	17.6.07	1	19981	+	-	-	+	-	-	-	-	+	+	+	-	-	106	R	S	AWMI	ST	+	+	-	-
71	Mohan Kumar	52	M	18.6.07	2	20033	+	-	-	-	-	-	-	-	+	+	-	-	-	36	R	IS	ASMI	CHB	-	+	TP	-
72	Nagarathinam	60	F	23.6.07	½	20184	+	-	-	-		-	-	-	+	-	+	+	-	112	R	S	ASMI	BFB	+	+	-	-
73	Mehboob	76	M	25.6.07	1	20891	+	-	-	-	+	-	-	-	+	-	-	-	-	140	IR	S	AWMI	LBBB	+	-	-	-
74	Arjunan	75	M	28.6.07	½	20938	-	-	+	+	-	-	-	+	-	-	-	-	-	106	R	S	ASMI	VT	+	-	+	+
75	Chandran	55	M	28.6.07	2	21479	-	+	-	-	-	-	+	-	-	+	+	-	-	80	R	S	ASMI	-	+	+	-	-
76	Chinnasamy	45	M	30.6.07	1	21485	+	-	+	-	+	+	-	+	+	+	+	-	-	36	IR	IS	ASMI	CHB	+	+	TP	-
77	Suresh	44	M	1.7.07	2	21616	-	+	-	+	-	-	+	-	-	+	+	-	-	130	R	S	AWMI	AF	+	+	+	-
78	Natarajan	36	M	1.7.07	2	21714	+	-	-	-	-	-	-	-	+	+	-	-	-	88	IR	S	ASMI	-	+	+	-	-
79	Abiyan	38	M	4.7.07	1	21719	+	-	-	-	-	-	-	-	+	+	-	-	-	90	IR	S	ASMI	-	+	+	-	-
80	Marimuthu	85	M	9.7.07	3	22123	+	-	-	+	-	-	-	-	+	+	+	-	-	84	R	S	EAWMI	BFB	+	+	-	-

81	Radhakrishnan	80	M	11.7.07	4	22893	+	+	+	-	-	-	+	+	+	+	-	-	-	78	R	S	EAWMI	VPD	+	-	-	-
82	Chandrasekar	50	M	14.7.07	3	23058	+	-	-	-	-	-	-	-	+	+	+	-	-	70	R	S	AWMI	BFB	+	+	-	-
83	Maniammal	60	F	15.7.07	½	23510	+	-	-	-	+	-	-	-	+	-	-	+	-	68	R	S	AWMI	VT	+	-	+	+
84	Ellammal	65	F	25.7.07	2	23564	+	+	-	-	-	-	+	-	+	-	-	+	-	90	R	S	AWMI	RBBB	+	-	-	-
85	Muthu Mary	45	F	27.7.07	2	24890	+	-	+	+	-	-	-	+	+	-	-	-	-	92	R	S	AWMI	-	+	+	-	-
86	Mannicbasha	81	M	31.7.07	2	25284	+	-	+	-	-	-	-	+	+	+	+	-	-	90	R	S	ASMI	RBBB	+	+	-	-
87	Poonusamy	56	M	1.8.07	1	25249	+	-	-	+	+	-	-	-	+	+	-	-	-	112	R	IS	AWMI	VF	+	-	+	+
88	Nithyananthan	44	M	1.8.07	½	25755	+	-	-	-	-	-	-	-	+	+	-	-	-	106	R	IS	AWMI	VT	+	-	+	+
89	Raghupathy	70	M	2.8.07	1	25804	+	-	-	-	-	-	-	-	+	+	+	-	-	124	R	S	ASMI	VT	-	+	+	+
90	Shankar	60	M	2.8.07	1	25967	+	-	-	+	-	-	-	-	+	+	+	-	-	112	R	S	AWMI	ST	-	-	-	-
91	Susheela	80	F	9.8.07	1	25983	+	-	-	+	-	-	-	-	+	-	-	+	-	94	R	IS	AWMI	BFB	-	-	-	-
92	Dilip Kumar	28	M	17.8.07	2	26872	+	-	-	-	-	-	-	-	+	+	-	-	-	106	R	IS	ASMI	-	+	+	-	-
93	Abdul Wakan	60	M	21.8.07	3	27855	+	-	-	-		-	-	-	+	+	+	-	-	90	R	S	ASMI	-	+	+	-	-
94	Kuppan	55	M	21.8.07	3	28264	+	-	-	-	+	+	-	-	+	+	-	-	-	88	R	S	AWMI	VT	+	+	+	+
95	Thambiraj	70	M	6.9.07	3	30088	-	-	+	+	-	-	-	+	-	+	+	-	-	40	R	IS	AWMI	CHB	+	+	TP	-
96	Selvan	64	M	7.9.07	4	30223	-	+	-	-	-	-	+	-	-	+	+	-	-	76	R	IS	AWMI	BFB	+	+	-	-
97	Susheela	67	F	8.9.07	2	40443	+	-	+	-	+	+	-	+	+	-	-	+	-	90	IR	S	ALMI	-	+	+	-	-
98	Thavarammal	75	F	9.9.07	6	30539	-	+	-	+	-	-	+	-	-	-	-	+	-	96	R	S	AWMI	-	+	-	-	-
99	Saraswathy	60	F	10.9.07	8	30686	+	-	-	-	-	+	-	-	+	-	-	+	-	94	R	S	ASMI	-	+	+	-	-
100	Mani	52	M	12.9.07	1	30879	+	-	-	-	-	+	-	-	+	+	+	-	-	130	R	S	AWMI	VT	+	-	+	+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